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 NEWS 1      Web Page URLs for STN Seminar Schedule - N. America
 NEWS 2  Sep 17  IMSworld Pharmaceutical Company Directory name change
         to PHARMASEARCH
 NEWS 3  Oct 09  Korean abstracts now included in Derwent World Patents
         Index
 NEWS 4  Oct 09  Number of Derwent World Patents Index updates increased
 NEWS 5  Oct 15  Calculated properties now in the REGISTRY/ZREGISTRY File
 NEWS 6  Oct 22  Over 1 million reactions added to CASREACT
 NEWS 7  Oct 22  DGENE GETSIM has been improved
 NEWS 8  Oct 29  AASD no longer available
 NEWS 9  Nov 19  New Search Capabilities USPATFULL and USPAT2
 NEWS 10 Nov 19  TOXCENTER(SM) - new toxicology file now available on STN
 NEWS 11 Nov 29  COPPERLIT now available on STN
 NEWS 12 Nov 29  DWPI revisions to NTIS and US Provisional Numbers
 NEWS 13 Nov 30  Files VETU and VETB to have open access
 NEWS 14 Dec 10  WPINDEX/WPIDS/WPIX New and Revised Manual Codes for
2002
 NEWS 15 Dec 10  DGENE BLAST Homology Search
 NEWS 16 Dec 17  WELDASEARCH now available on STN
 NEWS 17 Dec 17  STANDARDS now available on STN
 NEWS 18 Dec 17  New fields for DPCI
 NEWS 19 Dec 19  CAS Roles modified
 NEWS 20 Dec 19  1907-1946 data and page images added to CA and CAplus
 NEWS 21 Jan 25  BLAST(R) searching in REGISTRY available in STN on the
Web
 NEWS 22 Jan 25  Searching with the P indicator for Preparations
 NEWS 23 Jan 29  FSTA has been reloaded and moves to weekly updates
 NEWS 24 Feb 01  DKILIT now produced by FIZ Karlsruhe and has a new update
         frequency
 NEWS 25 Feb 19  Access via Tymnet and SprintNet Eliminated Effective 3/31/02

 NEWS EXPRESS  February 1 CURRENT WINDOWS VERSION IS V6.0d,
         CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP),
         AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
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=> s GPCR (3a) lympho
L1          0 GPCR (3A) LYMPHO

=> s GPCR (3a) lympho?
L2          5 GPCR (3A) LYMPHO?

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3          3 DUP REM L2 (2 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):y

L3  ANSWER 1 OF 3  CAPLUS  COPYRIGHT 2002 ACS
AN  2000:175932 CAPLUS
DN  132:218009
TI  Protein and cDNA sequences encoding G protein-coupled receptor 14275,
    which is related to the EDG receptor family, and uses thereof in drug
    screening, diagnostic, and therapeutic applications
IN  Glucksmann, Maria Alexandra; Hodge, Martin R.
PA  Millennium Pharmaceuticals, Inc., USA
SO  PCT Int. Appl., 117 pp.
    CODEN: PIXXD2
DT  Patent
LA  English
FAN.CNT 1
    PATENT NO.      KIND  DATE          APPLICATION NO.  DATE
    --------------- ----  --------      ---------------  --------
PI  WO 2000014233   A1    20000316      WO 1999-US20347  19990903
      W: AE, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR,
         CU, CZ, CZ, DE, DE, DK, DK, DM, EE, EE, ES, FI, FI, GB, GD, GE,
         GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK,
         LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO,
         RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN,
         YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
      RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK,
         ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG,
         CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
    AU 9962434      A1    20000327      AU 1999-62434    19990903
PRAI US 1998-146416  A    19980903
    US 1999-390039   A    19990903
    WO 1999-US20347  W    19990903
AB  The invention provides protein and cDNA sequences encoding a novel G
    protein-coupled receptor (14275), which is a new member of the EDG
    receptor family. The invention further relates to methods using receptor
    polypeptides and polynucleotides as a target for diagnosis and treatment
    in receptor-mediated disorders. The invention further relates to
    drug-screening methods using the receptor polypeptides and polynucleotides
    to identify agonists and antagonists for diagnosis and treatment. The
    invention further encompasses agonists and antagonists based on the
    receptor polypeptides and polynucleotides.
RE.CNT  4   THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS
RECORD
        ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3  ANSWER 2 OF 3  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS
INC.DUPLICATE 1
AN  2000:48924 BIOSIS
DN  PREV200000048924
TI  Kaposi's sarcoma-associated herpesvirus-encoded G protein-coupled receptor
    activation of c-Jun amino-terminal kinase/stress-activated protein kinase
    and Lyn kinase is mediated by related adhesion focal tyrosine
    kinase/proline-rich tyrosine kinase 2.
AU  Munshi, Neru; Ganju, Ramesh K.; Avraham, Shalom; Mesri, Enrique A.;
    Groopman, Jerome E. (1)
CS  (1) Division of Experimental Medicine and Hematology/Oncology, Harvard
    Institutes of Medicine-Beth Israel Deaconess Medical Center, 4 Blackfan
    Circle, Boston, MA USA
SO  Journal of Biological Chemistry, (Nov. 5, 1999) Vol. 274, No. 45, pp.
    31863-31867.
    ISSN: 0021-9258.
DT  Article
LA  English
SL  English
AB  The Kaposi's sarcoma-associated herpesvirus (KSHV) (also known as human
    herpesvirus 8) has been implicated in the pathogenesis of Kaposi's sarcoma
    and B cell primary effusion lymphomas. KSHV encodes a G protein-coupled
    receptor (GPCR) that acts as an oncogene and constitutively activates two
    protein kinases, c-Jun amino-terminal kinase (JNK)/stress-activated
    protein kinase (SAPK) and p38 mitogen-activated protein kinase. It also
    induces the production of vascular endothelial growth factor. These
    processes are believed to be important in KSHV-GPCR-related oncogenesis.
    We have characterized the signaling pathways mediated by KSHV-GPCR in a
    reconstituted 293T cell model in which the related adhesion focal tyrosine
    kinase (RAFTK) was ectopically expressed. RAFTK has been shown to play an
    important role in growth factor signaling in endothelium and in B cell
    antigen receptor signaling in B ***lymphocytes*** . KSHV- ***GPCR***
    induced the tyrosine phosphorylation of RAFTK. Expression of wild-type
    RAFTK enhanced GPCR-mediated JNK/SAPK activation, whereas
    dominant-negative mutant constructs of RAFTK, such as K457A (which lacks
    kinase activity) and Y402F (a Src-binding mutant), inhibited
    KSHV-GPCR-mediated activation of JNK/SAPK. RAFTK also mediated the
    KSHV-GPCR-induced activation of Lyn, a Src family kinase. However, RAFTK
    did not mediate the activation of p38 mitogen-activated protein kinase
    induced by KSHV-GPCR. Human interferon gamma-inducible protein-10, which
    is known to inhibit KSHV-GPCR activity, was found to reduce RAFTK
    phosphorylation and JNK/SAPK activation. These results suggest that in
    cells expressing RAFTK/proline-rich tyrosine kinase 2, such as endothelial
    and B cells, RAFTK can act to enhance KSHV-GPCR-mediated downstream
    signaling to transcriptional regulators such as JNK/SAPK.

L3  ANSWER 3 OF 3  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS
INC.DUPLICATE 2
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AN 1998:406091 BIOSIS
DN PREV199800406091
TI Human interferon-gamma-inducible protein 10 (IP-10) inhibits constitutive signaling of Kaposi's sarcoma-associated herpesvirus G protein-coupled receptor.
AU Geras-Raaka, Elizabeth; Varma, Anjali; Ho, Hao; Clark-Lewis, Ian; Gershengorn, Marvin C. (1)
CS (1) Cornell Univ. Med. Coll., 1300 York Ave., Rm. A328, New York, NY 10021 USA
SO Journal of Experimental Medicine, (July 20, 1998) Vol. 188, No. 2, pp. 405-408.
ISSN: 0022-1007.
DT Article
LA English
AB A G protein-coupled receptor (GPCR) is encoded within the genome of Kaposi's sarcoma-associated herpesvirus (KSHV)/human herpesvirus 8, a virus that may be involved in the pathogenesis of Kaposi's sarcoma and primary effusion ***lymphomas*** . KSHV- ***GPCR*** exhibits constitutive signaling activity that causes oncogenic transformation. We report that human interferon (IFN)-gamma-inducible protein 10 (HuIP-10), a C-X-C chemokine, specifically inhibits signaling of KSHV-GPCR. In contrast, monokine induced by IFN-gamma (HuMig), which like HuIP-10 is an agonist of C-X-C chemokine receptor 3, does not inhibit KSHV-GPCR signaling. Moreover, HuIP-10, but not HuMig, inhibits KSHV-GPCR-induced proliferation of NIH 3T3 cells. These results show that HuIP-10 is an inverse agonist that converts KSHV-GPCR from an active to an inactive state. Thus, a human chemokine inhibits constitutive signaling and cellular proliferation that is mediated by a receptor encoded by a human disease-associated herpesvirus.

=> s GPCR (5a) lympho?
L4 6 GPCR (5A) LYMPHO?

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5 4 DUP REM L4 (2 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 4 ANSWERS - CONTINUE? Y/(N):y

L5 ANSWER 1 OF 4 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2001:289395 BIOSIS
DN PREV200100289395
TI Refolding and isolation of an intact SLC/6Ckine expressed as an insoluble fusion protein in E coli.
AU Johanson, Kyung O. (1); Scott, Gilbert (1); McDevitt, Patrick (1); Matico, Rosalie (1); Sarau, Henry (1); Appelbaum, Edward (1)
CS (1) Protein Biochemistry, SmithKline Beecham Pharm, 709 Swedeland Rd, King of Prussia, PA, 19406 USA
SO FASEB Journal, (March 7, 2001) Vol. 15, No. 4, pp. A530. print.
Meeting Info.: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001
ISSN: 0892-6638.
DT Conference
LA English
SL English
AB SLC (secondary lymphoid tissue chemokine) is a C-C chemokine with three disulfide bonds (6Ckine) in 111 amino acids. It is a potent chemotactic factor for dendritic cells and T ***lymphocytes*** mediated through a 7TM ***GPCR*** receptor, CCR7. To investigate its potential as a drug target, various expression systems were tried in order to produce sufficient amount of an intact molecule. Natural N-terminus was determined using Drosophila secretion system using its own signal sequence, however the expression level was very low. The protein was then expressed at high level in E. coli as an insoluble protein with an N-terminal 6His tag with a cleavage site of enterokinase (EK) or factor Xa, or Genenasel. The inclusion body was solubilized and purified using NiNTA agarose in 6 M GdnHCl and reduced with DTT. GdnHCl and DTT were removed from the reduced/denatured chemokine by dialyzing against 5 mM HCl. It was then refolded by air oxidation at pH 8.5. EK or Xa treatment resulted in multiple cleavages at C-terminal region, while Genenase yielded an intact molecule. The chemokine was active in calcium mobilization assays with cells expressing recombinant CCR7 (EC50 = 1.2 nM).

L5 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2002 ACS
AN 2000:175932 CAPLUS
DN 132:218009
TI Protein and cDNA sequences encoding G protein-coupled receptor 14275, which is related to the EDG receptor family, and uses thereof in drug screening, diagnostic, and therapeutic applications
IN Glucksmann, Maria Alexandra; Hodge, Martin R.
PA Millennium Pharmaceuticals, Inc., USA
SO PCT Int. Appl., 117 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1
PATENT NO. KIND DATE APPLICATION NO. DATE
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PI WO 2000014233 A1 20000316 WO 1999-US20347 19990903
W: AE, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
AU 9962434 A1 20000327 AU 1999-62434 19990903
PRAI US 1998-146416 A 19980903
US 1999-390039 A 19990903
WO 1999-US20347 W 19990903
AB The invention provides protein and cDNA sequences encoding a novel G protein-coupled receptor (14275), which is a new member of the EDG receptor family. The invention further relates to methods using receptor polypeptides and polynucleotides as a target for diagnosis and treatment in receptor-mediated disorders. The invention further relates to drug-screening methods using the receptor polypeptides and polynucleotides to identify agonists and antagonists for diagnosis and treatment. The invention further encompasses agonists and antagonists based on the receptor polypeptides and polynucleotides.
RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 3 OF 4 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1
AN 2000:48924 BIOSIS
DN PREV200000048924
TI Kaposi's sarcoma-associated herpesvirus-encoded G protein-coupled receptor activation of c-Jun amino-terminal kinase/stress-activated protein kinase and Lyn kinase is mediated by related adhesion focal tyrosine kinase/proline-rich tyrosine kinase 2.
AU Munshi, Neru; Ganju, Ramesh K.; Avraham, Shalom; Mesri, Enrique A.; Groopman, Jerome E. (1)
CS (1) Division of Experimental Medicine and Hematology/Oncology, Harvard Institutes of Medicine-Beth Israel Deaconess Medical Center, 4 Blackfan Circle, Boston, MA USA
SO Journal of Biological Chemistry, (Nov. 5, 1999) Vol. 274, No. 45, pp. 31863-31867.
ISSN: 0021-9258.
DT Article
LA English
SL English
AB The Kaposi's sarcoma-associated herpesvirus (KSHV) (also known as human herpesvirus 8) has been implicated in the pathogenesis of Kaposi's sarcoma and B cell primary effusion lymphomas. KSHV encodes a G protein-coupled receptor (GPCR) that acts as an oncogene and constitutively activates two protein kinases, c-Jun amino-terminal kinase (JNK)/stress-activated protein kinase (SAPK) and p38 mitogen-activated protein kinase. It also induces the production of vascular endothelial growth factor. These processes are believed to be important in KSHV-GPCR-related oncogenesis. We have characterized the signaling pathways mediated by KSHV-GPCR in a reconstituted 293T cell model in which the related adhesion focal tyrosine kinase (RAFTK) was ectopically expressed. RAFTK has been shown to play an important role in growth factor signaling in endothelium and in B cell antigen receptor signaling in B ***lymphocytes*** . KSHV- ***GPCR*** induced the tyrosine phosphorylation of RAFTK. Expression of wild-type RAFTK enhanced GPCR-mediated JNK/SAPK activation, whereas dominant-negative mutant constructs of RAFTK, such as K457A (which lacks kinase activity) and Y402F (a Src-binding mutant), inhibited KSHV-GPCR-mediated activation of JNK/SAPK. RAFTK also mediated the KSHV-GPCR-induced activation of Lyn, a Src family kinase. However, RAFTK did not mediate the activation of p38 mitogen-activated protein kinase induced by KSHV-GPCR. Human interferon gamma-inducible protein-10, which is known to inhibit KSHV-GPCR activity, was found to reduce RAFTK phosphorylation and JNK/SAPK activation. These results suggest that in cells expressing RAFTK/proline-rich tyrosine kinase 2, such as endothelial and B cells, RAFTK can act to enhance KSHV-GPCR-mediated downstream signaling to transcriptional regulators such as JNK/SAPK.

L5 ANSWER 4 OF 4 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 2
AN 1998:406091 BIOSIS
DN PREV199800406091
TI Human interferon-gamma-inducible protein 10 (IP-10) inhibits constitutive signaling of Kaposi's sarcoma-associated herpesvirus G protein-coupled receptor.
AU Geras-Raaka, Elizabeth; Varma, Anjali; Ho, Hao; Clark-Lewis, Ian; Gershengorn, Marvin C. (1)
CS (1) Cornell Univ. Med. Coll., 1300 York Ave., Rm. A328, New York, NY 10021 USA
SO Journal of Experimental Medicine, (July 20, 1998) Vol. 188, No. 2, pp. 405-408.
ISSN: 0022-1007.
DT Article
LA English
AB A G protein-coupled receptor (GPCR) is encoded within the genome of Kaposi's sarcoma-associated herpesvirus (KSHV)/human herpesvirus 8, a virus that may be involved in the pathogenesis of Kaposi's sarcoma and primary effusion ***lymphomas*** . KSHV- ***GPCR*** exhibits

constitutive signaling activity that causes oncogenic transformation. We report that human interferon (IFN)-gamma-inducible protein 10 (HuIP-10), a C-X-C chemokine, specifically inhibits signaling of KSHV-GPCR. In contrast, monokine induced by IFN-gamma (HuMig), which like HuIP-10 is an agonist of C-X-C chemokine receptor 3, does not inhibit KSHV-GPCR signaling. Moreover, HuIP-10, but not HuMig, inhibits KSHV-GPCR-induced proliferation of NIH 3T3 cells. These results show that HuIP-10 is an inverse agonist that converts KSHV-GPCR from an active to an inactive state. Thus, a human chemokine inhibits constitutive signaling and cellular proliferation that is mediated by a receptor encoded by a human disease-associated herpesvirus.

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